Case Report

Acute Anuric Renal Failure Following Jering Bean Ingestion

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Djenkol beans or jering (*Pithecellobium jeringa*) is a traditional delicacy consumed by the local population in Malaysia. Jering poisoning or djenkolism is characterized by spasmodic pain, urinary obstruction and acute renal failure. The underlying pathology is an obstructive nephropathy, which is usually responsive to aggressive hydration and diuretic therapy. We present a case of djenkolism following ingestion of jering. The patient required urgent bilateral ureteric stenting following the failure of conservative therapy. Healthcare providers need to recognize djenkolism as a cause of acute renal failure and the public educated on this potential health hazard. [*Asian J Surg* 2007;30(1):80–1]

Key Words: acute renal failure, djenkolism, jering poisoning

Introduction

Djenkol bean or jering (*Pithecellobium jeringa*) is a traditional local delicacy eaten with the staple diet, rice (Figure).[1,2] It is eaten raw or half boiled, and locals believe that it has medicinal value.[2] Jering contains 1–2% djenkolic acid, a sulfur-containing amino acid.[3] Djenkolism or jering poisoning has been shown to cause mild to severe acute tubular obstruction with some glomerular cell necrosis.[3] Cases of jering nephropathy have been reported sporadically.[4–8]

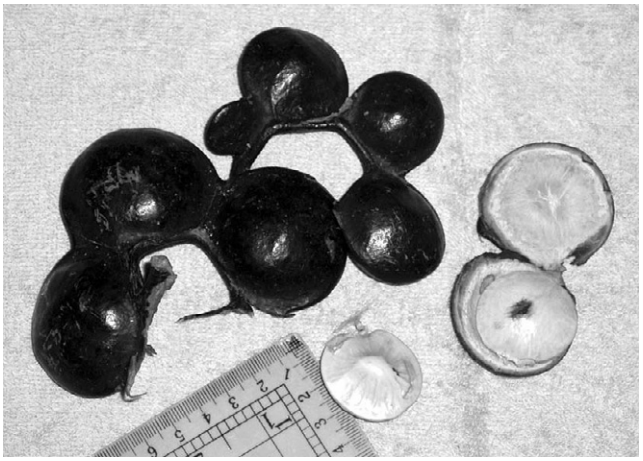

Figure. Jering beans.

The urine and breath of the affected individual usually have a pungent smell. This is a useful clue to the possible aetiology. Djenkolism varies in severity and presentation ranges from asymptomatic microhaematuria, mild abdominal colic, nausea, diarrhoea, constipation, dysuria to frank haematuria, severe loin or suprapubic pain and oligoanuric acute renal failure.[1,4,5]

Case report

A healthy 45-year-old man presented with colicky left loin pain, dysuria, frank haematuria and foul smelling urine a day after ingesting jering. He developed oliguria and was anuric by the 3rd day, when he was referred to our institution. When seen, he was normotensive and afebrile. Systemic

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examination was unremarkable. The urine showed frank haematuria.

The serum creatinine was 176 μmol/L, serum urea 18 mmol/L, potassium 4.4 mmol/L and bicarbonate 21.1 mmol/L. Haemoglobin was 14.1 g/dL, total white cell count 12.8×10^9/L, platelets 177×10^9/L and his coagulation profile was normal. Ultrasound of the kidneys showed normal sized kidneys with minimal right hydronephrosis.

Initially, he was treated with intravenous hydration and frusemide, but urine output remained poor. Serum creatinine increased from 176 to 848 μmol/L over 3 days. Hence, urgent bilateral ureteric stenting was done, and thick "tomato sauce" sludge was found. The patient subsequently had good diuresis after the procedure. The stents were removed 4 days later. He made an uneventful recovery and serum creatinine on discharge was 79 μmol/L.

Discussion

This case illustrates the need to consider djenkolism as a cause of acute anuric renal failure. Djenkolism occurs within 48 hours of consumption of jering. Precipitation of the djenkolic acid in urine produces viscous urine forming sludge. This may progress to obstructive nephropathy leading to acute tubular necrosis.[5] Conservative treatment is with aggressive hydration and alkalinization of the urine to increase the solubility of djenkolic acid. A high urine flow is needed to flush out the sludge.[5–7] However, some cases of severe djenkolism with anuria do not respond to conservative therapy. Prompt surgical intervention needs to be considered in this situation, but there is no consensus on which is the best treatment option. Our patient had bilateral ureteric stent insertion. It is likely that simple flushing of both the ureters would be just as effective. Once the obstruction is relieved and a good flow of urine is established, the patient is expected to recover with no residual renal impairment.

As jering or djenkol bean is consumed widely in this region, efforts to increase awareness of this potential health hazard of jering are needed.

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